# EM CASES SUMMARY

## Episode 155 Treatment of Bradycardia and Bradydysrhythmias

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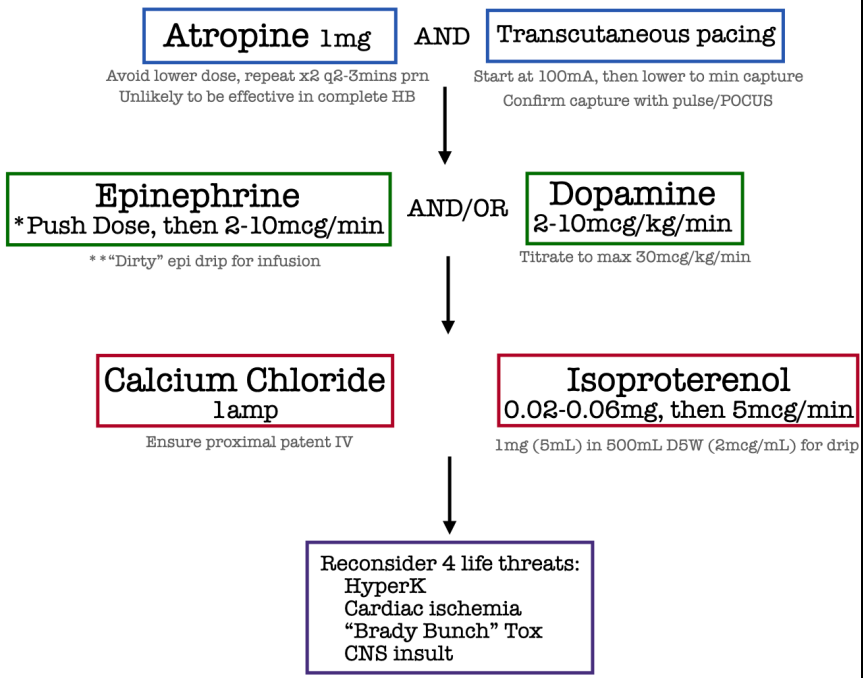


- For the crashing patient administer drugs/fluids and start transcutaneous pacing simultaneously
- There is a paucity of randomized trials for medications to treat bradycardia; these recommendations are based on the ACLS guidelines, weak observational data and expert opinion
- The underlying cause needs to be taken into consideration (i.e., for B-blocker overdose or Calcium channel blocker overdose, consider high dose insulin, for digoxin overdose consider digiFab, for the hypothermic patient rewarming is usually required before consideration of medications or pacing, for myxedema coma, consider thyroxine)

## Atropine for bradycardia and bradydysrhythmias

Atropine essentially fires up the SA node by poisoning the vagus nerve; it therefore is effective only if the distal conduction system is conducting normally. Overall, only 28% of patients with bradycardia have been shown to respond to atropine.

Appropriately dosed atropine is usually effective for **proximal AV block, sinus bradycardia and junctional rhythms** but is not useful (nor particularly harmful) in distal AV block (idioventricular rhythms and second-degree type II and third-degree AV block).

In the latest 2020 AHA update the recommended single dose administration of atropine was increased from 0.5 mg to 1 mg based on data suggesting that at low doses, atropine may cause paradoxical bradycardia. At low doses, atropine *decreases* heart rate by blocking M1 acetylcholine receptors in the parasympathetic ganglion controlling the SA node. At higher doses, atropine *increases* heart rate by blocking M2 acetylcholine receptors on the myocardium itself. Atropine-induced bradycardia may be especially difficult to manage in patients who are morbidly obese or post cardiac transplantation.

Atropine dosing: 1mg IV q3 mins, max 3mg (cholinergic poisoning may require higher doses using a doubling approach: 1mg, then 2mg, 4mg, 8mg etc.)

***Beware:*** *cardiac transplant patients may have paradoxical worsening of bradycardia with atropine; atropine should generally be avoided in cardiac transplant patients*
Most sick bradycardia patients will *not* respond to atropine, so it is important to move quickly to chronotropic drugs such as epinephrine if atropine is not effective.

***Pitfall:*** *underdosing atropine and waiting for it to work too long before moving onto a chronotropic drug is a common pitfall in the treatment of bradycardia*

## Epinephrine and dopamine for bradycardia and bradydysrhythmias

If the first dose of 1mg atropine IV is ineffective, move quickly to chronotropic drugs – dopamine and/or epinephrine – while concurrently administering additional 1mg doses of atropine q3 mins (max 3mg). The advantage of dopamine is that it comes premixed and so can be started quickly, while epinephrine push-dose or infusion both require mixing.

Epinephrine dosing: 2-10 mcg/min infusion (it is safe to use a proximal peripheral line initially with frequent limb checks)
Temporize with push dose epinephrine boluses of ~20-50 mcg

Source: Internet Book of Critical Care, Jan, 2017 Josh Farkas

## Calcium for bradycardia and bradydysrhythmias

If atropine, dopamine, epinephrine and pacing are ineffective, and the cause of bradycardia is unclear, consider IV calcium chloride or calcium gluconate.
Calcium-responsive bradycardias

- Hyperkalemia
- Hypocalcemia
- Hypermagnesemia
- Calcium-channel blocker overdose

Dosing: Calcium chloride 1g OR Calcium gluconate 3g

## Reverse any other underlying cause of bradycardia

Cardiac ischemia – primary goal is cardiac catheterization/revascularization; transfer patient to cath lab ASAP, consider bradycardia medications/transcutaneous pacing only as a bridge to catheterization; use minimal doses of dopamine and/or epinephrine as either drug may exacerbate cardiac ischemia

Hypothermia – warming measures usually preclude the need for bradycardia medications/pacing; rewarming is the first line treatment for bradycardia in patients with severe hypothermia; pacing may precipitate ventricular fibrillation in severely hypothermic patients

Myxedema coma – thyroxine

*Pearl: inferior MIs tend to cause narrow complex, transient, vagal-type bradycardia that is likely to respond to atropine vs anterior MIs tend to cause wide complex bradydysrhythmias that require pacing*

## Pearls on treatment of "The Brady Bunch" toxicologic causes of bradycardia

- Pacing is unlikely to be successful in B-blocker and Ca-blocker poisonings
- For B-blocker and Ca-blocker consider high dose insulin and lipid emulsion
- Glucagon should be reserved as a last resort and should not be given routinely
- Digoxin toxicity can cause almost any bradyarrhythmia from junctional bradycardia to complete heart block
- One clue to differentiate calcium blocker toxicity from B-blocker toxicity is that Ca-blocker poisoning tends to cause hyperglycemia vs B-blocker poisoning tends to be normoglycemic or hypoglycemic
- Consider bicarb for propranolol toxicity as it has NA channel blocking properties; QRS widens as the toxic effects progress

## Transcutaneous pacing for bradycardia and bradydysrhythmias

In the crashing bradycardic patient, transcutaneous pacing should be started in parallel with medications. Transcutaneous pacing is a temporizing measure to definite care in the crashing bradycardia patient; when one has more than a few minutes to correct the bradycardia, transvenous pacing is preferred.

## Pitfalls in transcutaneous pacing

- When employing transcutaneous or transvenous pacing, confirm real capture that results in a ventricular beat with femoral pulse checks (ideally using POCUS) and pulse oximetry wave. Do not rely solely on the monitor/ECG.
- Do not use a carotid pulse check for the assessment of circulation as TCP can create muscular movements that may feel like a carotid pulse. Assess circulation using the femoral pulse (with POCUS ideally).
- If the patient is crashing start the output high (i.e.,100mA); do not start at 5mA and increase as one would for more stable patients

### Setting the output and on the pacer

- In the crashing patient, start at 100mA and titrate downward to 5-20mA above the minimum energy required for capture; if not capturing, increase to max 130mA and if still not capturing, move the pads to improve the vector through the heart and try again.
- Patients with obesity and COPD typically require ~40-80 mA more than other patients to capture
- Our experts suggest to set the rate of pacing at 60 bpm

Consider ketamine as your first line analgesic for the patient undergoing transcutaneous pacing as it is least likely to cause hypotension, may help increase the heart rate and it helps maintains respirations

## Transvenous pacing for bradycardia and bradydysrhythmias

- Transvenous is much more effective than transcutaneous pacing with success rates of >95%
- Transvenous pacing is indicated when drugs fail and for high degree AV blocks
- Start at 5mA output in stable patients and titrate upwards until capture; then set output at 5-20mA above the minimal capture output
- Use POCUS to follow the wire to make sure the tip sits in the IVC

## Indications for permanent pacemaker for bradycardia and bradydysrhythmias

The decision to place a permanent pacemaker is almost never made in the ED; nonetheless it is important for the EM physician to be aware of which patients are likely to require permanent pacemaker at a later date.

The AHA guidelines state: "In sinus node dysfunction, there is no established minimum heart rate or pause duration where permanent pacing is recommended. Establishing temporal correlation between symptoms and bradycardia is important when determining whether permanent pacing is needed. In patients with acquired second-degree Mobitz type II atrioventricular block, high-grade atrioventricular block, or third-degree atrioventricular block not caused by reversible or physiologic causes, permanent pacing is recommended regardless of symptoms. For all other types of atrioventricular block, in the absence of conditions associated with progressive atrioventricular conduction abnormalities, permanent pacing should generally be considered only in the presence of symptoms that correlate with atrioventricular block."

Dr. Dorian has found in practice that patients with sinus node disease or with AV nodal block not infrequently inappropriately receive pacemakers, while patients with bundle branch block with seemingly innocuous syncope who should receive a permanent pacemaker, sometimes do not, even though they may be at risk for life-threatening distal block.

## REFERENCES


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